L Number	Hits	Search Text	DB	Time stamp
1	40	mirabelli$.in.	USPAT; US-PGPUB	2003/01/15 07:35
2	10610	viral near5 (growth or replication)	USPAT; US-PGPUB	2003/01/15 07:36
3	16103	(virus or viral) near5 (growth or replication)	USPAT; US-PGPUB	2003/01/15 07:36
4	1827	proliferation near5 gene	USPAT; US-PGPUB	2003/01/15 07:37
5	31	((virus or viral) near5 (growth or replication)) same (proliferation near5 gene)	USPAT; US-PGPUB	2003/01/15 07:43
6	5869	((virus or viral) near5 (growth or replication)) same host	USPAT; US-PGPUB	2003/01/15 07:44
7	29	((virus or viral) near5 (growth or replication)) same host near5 proliferat$5	USPAT; US-PGPUB	2003/01/15 07:47
8	23	((virus or viral) near5 (growth or replication)) same screen$4 near4 host	USPAT; US-PGPUB	2003/01/15 07:51

FILE 'HOME' ENTERED AT 07:37:24 ON 15 JAN 2003

=> file medline biosis caplus
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.78	3.78

FILE 'MEDLINE' ENTERED AT 07:47:58 ON 15 JAN 2003

FILE 'BIOSIS' ENTERED AT 07:47:58 ON 15 JAN 2003
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FILE 'CAPLUS' ENTERED AT 07:47:58 ON 15 JAN 2003
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=> (virus or viral)
(VIRUS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s (virus or viral)
L1 1263570 (VIRUS OR VIRAL)

=> s (growth or replicat? or proliferat?)
L2 3204498 (GROWTH OR REPLICAT? OR PROLIFERAT?)

=> s l1 (7a) l2
L3 117769 L1 (7A) L2

=> s l3 and (screen? (5a) host (3a) cell?)
L4 10 L3 AND (SCREEN? (5A) HOST (3A) CELL?)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 8 DUP REM L4 (2 DUPLICATES REMOVED)

=> d 1-8 bib ab

L5 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:423718 BIOSIS
DN PREV200200423718
TI Methods to assay gene function with viral vectors.
AU Dropulic, Boro; Pitha-Rowe, Paula (1)
CS (1) Baltimore, MD USA
ASSIGNEE: The Johns Hopkins University School of Medicine
PI US 6410257 June 25, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (June 25, 2002) Vol. 1259, No. 4, pp. No Pagination. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133.
DT Patent
LA English
AB The present invention provides a conditionally **replicating** **viral** vector, methods of making, modifying, propagating and selectively packaging, and using such a vector, isolated molecules of specified nucleotide and amino acid sequences relevant to such vectors, a pharmaceutical composition and a host cell comprising such a vector, the

use of such a **host cell** to **screen** drugs. The methods include the prophylactic and therapeutic treatment of viral infection, in particular HIV infection, and, thus, are also directed to vital vaccines and the treatment of cancer, in particular cancer of viral etiology. Other methods include the use of such conditionally **replicating viral** vectors in gene therapy and other applications.

L5 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2003 ACS
AN 2002:777654 CAPLUS
DN 137:289918
TI Improved conditionally **replicating** lentivirus vectors inhibiting wild-type **virus replication** and their therapeutic uses
IN Humeau, Laurent; Li, Yuexia; Merling, Randall; Dropulic, Boro; Schonely, Kathy L.
PA Virxsys, USA
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002078631	A2	20021010	WO 2002-US9526	20020326

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-819401 A 20010327
AB The present invention provides improved conditionally replicating vectors that have improved safety against the generation of **replication** competent vectors or **virus**. The vector is dependent upon an external agent to replicate in a target cell, such as a cell infected by the wild-type virus. The agent may be a gene deleted in the vector but not from the wild-type **virus**, making it dependent upon infection for **replication**. The vector carries a gene for an agent that gives the vector a selective advantage over the wild-type virus in the target cells, such as a ribozyme specific to the wild type virus. As the wild-type **virus** is eliminated, the vector stops **replicating** and is therefore at a lower risk of recombining with wild-type virus. Also disclosed are methods of making, propagating and selectively packaging, modifying and using vectors. Included are improved helper constructs, host cells, for use with the improved vectors as well as pharmaceutical compns. and host cells comprising the vectors, the use of vector contg. **host cells** to **screen** drugs, and methods of using the vectors to det. gene function. The methods also include the prophylactic and therapeutic treatment of disease, esp. viral infection, and HIV infection in particular. The development of a vector based on HIV-1 carrying a ribozyme against the U5 element is demonstrated. The vector is made resistant to the ribozyme by changes in the sequence of its U5 element.

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2003 ACS
AN 2002:240958 CAPLUS
DN 136:274271
TI Non-infective **viral** vectors for therapeutic use that can block **replication** of of wild-type **virus**

IN Chang, Yung-Nien; Lu, Xiaobin; Slepushkin, Vladimir; Conde, Betty; Davis, Brian; Yu, Qiao; Yang, Yanping; Merling, Randal; Han, Wei; Ni, Yajin; Li, Yuexia; Dropulic, Boro
PA Virxsys, USA
SO PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002024897	A2	20020328	WO 2001-US29976	20010921
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2001093075	A5	20020402	AU 2001-93075	20010921
PRAI	US 2000-667893	A	20000922		
	WO 2001-US29976	W	20010921		

AB Viral vectors for therapeutic use that lack genes essential for replication in a target cell and that can inhibit the **replication** of a wild-type **virus** that may arise by recombination are described. The vectors carry a gene for a ribozyme or antisense nucleic acid that will act on a sequence found only in the **replication** -competent **virus** and block its **replication**. Also disclosed are methods of making, propagating and selectively packaging, modifying, and using such vectors. Included are improved helper constructs, host cells, for use with the improved vectors as well as pharmaceutical compns. and host cells comprising the vectors, the use of vector contg. **host cells** to **screen** drugs, and methods of using the vectors to det. gene function. The methods also include the prophylactic and therapeutic treatment of disease, esp. viral infection, and HIV infection in particular. The construction of an HIV-1-based vector that included a gene for a hammerhead ribozyme directed against the U5 region of wild-type HIV-1 is described. The U5 region of the vector was modified to resist ribozyme cleavage.

L5 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:203639 BIOSIS
DN PREV200100203639
TI Methods to express genes from viral vectors.
AU Dropulic, Boro (1); Pitha, Paula M.
CS (1) Ellicott City, MD USA
ASSIGNEE: The Johns Hopkins University School of Medicine
PI US 6114141 September 05, 2000
SO Official Gazette of the United States Patent and Trademark Office Patents, (Sep. 5, 2000) Vol. 1238, No. 1, pp. No Pagination. e-file.
ISSN: 0098-1133.
DT Patent
LA English
AB The present invention provides a conditionally **replicating** **viral** vector, methods of making, modifying, propagating and selectively packaging, and using such a vector, isolated molecules of specified nucleotide and amino acid sequences relevant to such vectors, a pharmaceutical composition and a host cell comprising such a vector, the use of such a **host cell** to **screen** drugs. The methods include the prophylactic and therapeutic treatment of viral

infection, in particular HIV infection, and, thus, are also directed to viral vaccines and the treatment of cancer, in particular cancer of viral etiology. Other methods include the use of such conditionally **replicating viral** vectors in gene therapy and other applications.

L5 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2003 ACS
AN 1999:205258 CAPLUS
DN 130:233260
TI Conditionally **replicating viral** vectors and their use in vaccines, **viral** infection treatment, or cancer therapy
IN Dropulic, Boro; Pitha, Paula M.
PA The Johns Hopkins University School of Medicine, USA
SO U.S., 31 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5885806	A	19990323	US 1996-758598	19961127
	US 5888767	A	19990330	US 1997-917625	19970822
	US 6114141	A	20000905	US 1999-251085	19990216
	US 6207426	B1	20010327	US 1999-251084	19990216
	US 6232120	B1	20010515	US 1999-251283	19990216
	US 6168953	B1	20010102	US 1999-312322	19990514
	US 6498033	B1	20021224	US 2000-524004	20000313
	US 6410257	B1	20020625	US 2000-562894	20000501
PRAI	US 1993-32800P	P	19931128		
	US 1995-32800P	P	19951128		
	US 1996-758598	A3	19961127		
	US 1997-917625	A3	19970822		
	US 1999-251085	A3	19990216		
	US 1999-251283	A3	19990216		

AB The present invention provides a conditionally **replicating viral** vector, methods of making, modifying, propagating and selectively packaging, and using such a vector, isolated mols. of specified nucleotide and amino acid sequences relevant to such vectors, a pharmaceutical compn. and a host cell comprising such a vector, and the use of such a **host cell** to **screen** drugs.
The methods include the prophylactic and therapeutic treatment of viral infection, in particular HIV infection, and, thus, are also directed to viral vaccines and the treatment of cancer, in particular cancer of viral etiol. Other methods include the use of such conditionally **replicating viral** vectors in gene therapy and other applications. Examples include conditionally replicating HIV vectors crHIV-1.1, crHIV-1.11, crHIV-1.12, and crHIV-1.111. Examples also include use of triple anti-TAT ribozyme cassettes to cleave HIV nucleic acids.
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2003 ACS
AN 1997:457172 CAPLUS
DN 127:76993
TI Conditionally **replicating viral** vectors and their use in vaccines, **viral** infection treatment, or cancer therapy
IN Dropulic, Boro; Pitha, Paula M.
PA Johns Hopkins University School of Medicine, USA
SO PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DT Patent
LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9720060	A1	19970605	WO 1996-US18997	19961127
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9711249	A1	19970619	AU 1997-11249	19961127
	EP 871757	A1	19981021	EP 1996-942083	19961127
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	CN 1207775	A	19990210	CN 1996-199726	19961127
	JP 2000503527	T2	20000328	JP 1997-520658	19961127
	BR 9612574	A	20000425	BR 1996-12574	19961127
	NO 9802418	A	19980727	NO 1998-2418	19980527
PRAI	US 1995-563459	A	19951128		
	WO 1996-US18997	W	19961127		

AB The present invention provides a conditionally **replicating viral** vector, methods of making, modifying, propagating and selectively packaging, and using such a vector, isolated mols. of specified nucleotide and amino acid sequences relevant to such vectors, a pharmaceutical compn. and a host cell comprising such a vector, and the use of such a **host cell** to **screen** drugs.
The methods include the prophylactic and therapeutic treatment of viral infection, in particular HIV infection, and, thus, are also directed to viral vaccines and the treatment of cancer, in particular cancer of viral etiol. Other methods include the use of such conditionally **replicating viral** vectors in gene therapy and other applications. Examples include conditionally replicating HIV vectors crHIV-1.1, crHIV-1.11, crHIV-1.12, and crHIV-1.111. Examples also include use of triple anti-TAT ribozyme cassettes to cleave HIV nucleic acids.

L5 ANSWER 7 OF 8 MEDLINE DUPLICATE 1
AN 94233727 MEDLINE
DN 94233727 PubMed ID: 7513920
TI Identification and characterization of a murine cytomegalovirus gene with homology to the UL25 open reading frame of human cytomegalovirus.
AU Dallas P B; Lyons P A; Hudson J B; Scalzo A A; Shellam G R
CS Department of Microbiology, University of Western Australia, Nedlands.
SO VIROLOGY, (1994 May 1) 200 (2) 643-50.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U02500
EM 199406
ED Entered STN: 19940620
Last Updated on STN: 19960129
Entered Medline: 19940606
AB Monoclonal antibody 1B4, previously shown to be protective in vivo and to cross-react with both virally encoded and normal **host cell** proteins, was used to **screen** a lambda gt11 cDNA derived from mRNA harvested from mouse embryo fibroblasts 24 hr after infection with murine cytomegalovirus (MCMV). A 700-bp cDNA was identified representing the 5'terminus of a 2460-bp open reading frame (ORF) with significant homology to the human cytomegalovirus UL25 ORF. The UL25 ORF

of MCMV potentially encodes an 820 amino acid viral tegument protein with an estimated molecular weight of approximately 90 kDa. Amino acid homology with eukaryotic nucleolins was identified in the acidic N-terminal third of the MCMV UL25 proteins, suggesting that the protein may be involved in transcriptional activation or interactions with chromatin. Northern analysis and S1 nuclease data indicated that the gene is expressed late in infection as an approximately 3-kb transcript and that expression is dependent on **viral** DNA **replication**. An epitope recognized by MAb 1B4 was identified using recombinant pGEX plasmids expressing fusion proteins representing the N-terminal region of the MCMV UL25 protein. The identification of the MCMV UL25 ORF as a member of the CMV-specific UL25/UL35 gene family provides an opportunity for the investigation of the role these genes and their products in CMV pathogenesis in an animal model.

L5 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2003 ACS
AN 1990:30342 CAPLUS
DN 112:30342
TI Use of host cell reactivation of cisplatin-treated adenovirus 5 in human cell lines to detect repair of drug-treated DNA
AU Maynard, Kevin R.; Hosking, Louise K.; Hill, Bridget T.
CS Lab. Cell. Chemother., Imp. Cancer Res. Fund, London, WC2A 3PX, UK
SO Chemico-Biological Interactions (1989), 71(4), 353-65
CODEN: CBINA8; ISSN: 0009-2797
DT Journal
LA English
AB This study demonstrates that: (i) cisplatin (CDDP)-treated adenovirus can be used as a method for screening cell lines for some DNA-repair deficiencies. However, not all DNA-repair deficiencies affected reactivation of CdDP-treated adenovirus; (ii) there are at least 3 CDDP-DNA adducts formed which do not affect he replication of CDDP-treated Ad5 in the SuSa cell line relative to the **replication** of the **virus** in the RT112 cell line; (iii) it is possible to use CDDP-treated virus as a lethal probe to obtain CDDP-repair deficient cell lines.

=> d his

(FILE 'HOME' ENTERED AT 07:37:24 ON 15 JAN 2003)

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 07:47:58 ON 15 JAN 2003
L1 1263570 S (VIRUS OR VIRAL)
L2 3204498 S (GROWTH OR REPLICAT? OR PROLIFERAT?)
L3 117769 S L1 (7A) L2
L4 10 S L3 AND (SCREEN? (5A) HOST (3A) CELL?)
L5 8 DUP REM L4 (2 DUPLICATES REMOVED)

=> s l3 and (screen? (5a) host)
L6 21 L3 AND (SCREEN? (5A) HOST)

=> s l6 not l4
L7 11 L6 NOT L4

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 7 DUP REM L7 (4 DUPLICATES REMOVED)

=> d 1-7 bib ab

L8 ANSWER 1 OF 7 MEDLINE DUPLICATE 1
AN 2002114464 MEDLINE

DN 21835702 PubMed ID: 11847122
TI A chloroplast protein binds a viroid RNA in vivo and facilitates its hammerhead-mediated self-cleavage.
AU Daros Jose-Antonio; Flores Ricardo
CS Instituto de Biologia Molecular y Celular de Plantas (UPV-CSIC), Universidad Politecnica de Valencia, Avenida de los Naranjos s/n, Valencia 46022, Spain.
SO EMBO JOURNAL, (2002 Feb 15) 21 (4) 749-59.
Journal code: 8208664. ISSN: 0261-4189.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200204
ED Entered STN: 20020216
Last Updated on STN: 20020420
Entered Medline: 20020419
AB Viroids, small single-stranded circular RNAs (246-401 nucleotides), do not have mRNA capacity and must recruit host proteins to assist in the steps of their biological cycle. The nature of these cellular factors is poorly understood due to a lack of reliable experimental approaches. Here, to **screen** for **host** proteins interacting with viroid RNAs in vivo, we UV-irradiated avocado leaves infected with avocado sunblotch viroid (ASBVd), the type member of chloroplast viroids containing hammerhead ribozymes. This resulted in the detection of several ASBVd-host protein adducts. Tandem mass spectrometry analysis of the most abundant cross-linked species identified the protein component as two closely related chloroplast RNA-binding proteins (PARBP33 and PARBP35) of a family whose members previously have been shown to be involved in stabilization, maturation and editing of chloroplast transcripts. PARBP33 behaves as an RNA chaperone that stimulates in vitro the hammerhead-mediated self-cleavage of the multimeric ASBVd transcripts that result from rolling circle replication, indicating that this reaction, despite its RNA-based mechanism, is facilitated by proteins. The structural and functional parallelism between PARBP33 and PARBP35, and some proteins involved in **viral** RNA **replication**, indicates that viroids and RNA viruses recruit similar host proteins for their replication.

L8 ANSWER 2 OF 7 MEDLINE DUPLICATE 2
AN 2002294666 MEDLINE
DN 22031235 PubMed ID: 12033790
TI Silencing of a gene encoding a protein component of the oxygen-evolving complex of photosystem II enhances **virus replication** in plants.
AU Abbink Truus E M; Peart Jack R; Mos Thera N M; Baulcombe David C; Bol John F; Linthorst Huub J M
CS Institute of Molecular Plant Sciences, Gorlaeus Laboratories, Leiden University, 2300 RA, The Netherlands.
SO VIROLOGY, (2002 Apr 10) 295 (2) 307-19.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF426837
EM 200207
ED Entered STN: 20020530
Last Updated on STN: 20020713
Entered Medline: 20020712
AB It has been suggested that, in addition to viral proteins, host proteins are involved in RNA **virus replication**. In this study the RNA helicase domain of the Tobacco mosaic virus (TMV) replicase

proteins was used as bait in the yeast two-hybrid system to identify tobacco proteins with a putative role in TMV replication. Two host proteins were characterized. One protein (designated #3) belongs to a protein family of ATPases associated with various activities (AAA), while the second host protein (designated #13) is the 33K subunit of the oxygen-evolving complex of photosystem II. Using Tobacco rattle virus vectors, genes #3 and #13 were silenced in Nicotiana benthamiana, after which the plants were challenged by TMV infection. Silencing of gene #13 resulted in a 10-fold increase of TMV accumulation, whereas silencing of gene #3 caused a twofold reduction of TMV accumulation. Additionally, silencing of genes #3 and #13 decreased and increased, respectively, the accumulation of two other viruses. Similar to silencing of gene #13, inhibition of photosystem II by application of an herbicide increased TMV accumulation several fold. Infection of N. benthamiana with TMV resulted in a decrease of #13 mRNA levels. Silencing of gene #13 may reflect a novel strategy of TMV to suppress basal **host** defense mechanisms. The two-hybrid **screenings** did not identify tobacco proteins involved in helicase domain-induced N-mediated resistance.

L8 ANSWER 3 OF 7 CAPLUS
AN 2001:115376 CAPLUS
DN 134:173015
TI Identification and use of antiviral compounds that inhibit interaction of host cell proteins and **viral** proteins required for **viral replication**
IN O'Neill, Robert; Harty, Ronald; Palese, Peter M.
PA Mount Sinai School of Medicine of New York University, USA
SO PCT Int. Appl., 147 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001011335	A2	20010215	WO 2000-US22257	20000811
	WO 2001011335	C2	20020711		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2000067713	A5	20010305	AU 2000-67713	20000811
PRAI	US 1999-148263P	P	19990811		
	WO 2000-US22257	W	20000811		

OS MARPAT 134:173015
AB The present invention relates to the identification of host cell proteins that interact with **viral** proteins required for **virus replication**, and high throughput assays to identify compds. that interfere with the specific interaction between the viral and host cell protein. Interfering compds. that inhibit **viral replication** can be used therapeutically to treat **viral** infection. The invention is based, in part, on the Applicants' discovery of novel interactions between viral proteins and a human host cell protein. One of these host cell proteins, referred to herein as NPI-1, interacts with influenza virus protein NP. Also, host cell proteins, referred to herein as NS1I-1 and NS1-BP interact with influenza virus protein NS1. In addn., host cell proteins contg. WW domains that interact

with viral proteins such as Rhabdoviral M protein are described. Compds. that interfere with the binding of the host cell and **viral** proteins, and inhibit **viral replication** can be useful for treating **viral** infection in vivo.

L8 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2003 ACS
AN 1998:430038 CAPLUS
DN 129:76479
TI Screening method for the identification of compounds capable of abrogation HIV-1 Gag-cyclophilin complex formation
IN Luban, Jeremy; Goff, Stephen P.
PA Columbia University In the City of New York, USA
SO U.S., 22 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5773225	A	19980630	US 1994-248357	19940524
PRAI	US 1994-248357		19940524		

AB The human immunodeficiency virus type 1 (HIV-1) gag gene product is capable of directing the assembly of virion particles independent of other viral elements. The Gag protein also plays an important role during the early stages of **viral replication**. Employing the yeast two-hybrid system, a cDNA expression library was **screened** and two **host** proteins identified. These proteins, designated cyclophilins A and B (CyPsA and B), interacted specifically with the HIV-1 Gag polyprotein Pr55gag. Glutathione S-transferase-CyP fusion proteins bind tightly to Pr55gag in vitro. Cyclosporin A (CsA) efficiently disrupts the Gag-CyPA binding interaction. The identification of novel compds. capable of abrogating this protein-protein interaction employing the disclosed screening assay will facilitate the development of HIV-1 antiviral agents.

RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2003 ACS
AN 1997:332442 CAPLUS
DN 126:302368
TI Infected host proteins that interact with virus proteins, host protein cDNA sequences, and recombinant expression systems for antiviral compound screening
IN Palese, Peter; O'Neill, Robert
PA Mount Sinai Medical Center, USA
SO PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9712967	A1	19970410	WO 1995-US13044	19951006
	W: AL, AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IS, JP, KG, KP, KR, KZ, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TT, UA, UZ, VN				
	RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2234047	AA	19970410	CA 1995-2234047	19951006
	AU 9539538	A1	19970428	AU 1995-39538	19951006
	EP 861322	A1	19980902	EP 1995-937415	19951006

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE
JP 11513252 T2 19991116 JP 1996-514221 19951006
PRAI WO 1995-US13044 W 19951006
AB The present invention relates to the identification of host cell proteins that interact with **viral** proteins required for **virus replication**, and high throughput assays to identify compds. that interfere with the specific interaction between the viral and host cell protein. Interfering compds. that inhibit **viral replication** can be used therapeutically to treat **viral** infection. The invention is based, in part, on the Applicants' discovery of novel interactions between proteins of the influenza virus and human host cell proteins. One of these host cell proteins, referred to herein as NPI-1, interacts with influenza virus protein NP, and may be an accessory protein required for **replication** of influenza **virus**. Another of these host cell proteins, referred to herein as NS1I-1, interacts with influenza virus protein NS1. Compds. that interfere with the binding of the host cell and **viral** proteins, and inhibit **viral replication** can be useful for treating **viral** infection in vivo.

L8 ANSWER 6 OF 7 MEDLINE DUPLICATE 3
AN 95014038 MEDLINE
DN 95014038 PubMed ID: 7928964
TI DNA replication studies with coliphage 186: the involvement of the Escherichia coli DnaA protein in 186 replication is indirect.
AU Williams S G; Egan J B
CS Department of Biochemistry, University of Adelaide, Australia.
SO JOURNAL OF BACTERIOLOGY, (1994 Oct) 176 (19) 6039-44.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941028
AB The inability of coliphage 186 to infect productively a dnaA(Ts) mutant at a restrictive temperature was confirmed. However, the requirement by 186 for DnaA is indirect, since 186 can successfully infect suppressed dnaA (null) strains. The block to 186 infection of a dnaA(Ts) strain at a restrictive temperature is at the level of replication but incompletely so, since some 20% of the phage specific replication seen with infection of a dnaA+ **host** does occur. A mutant **screen**, to isolate **host** mutants blocked in 186-specific replication but not in the replication of the close relative coliphage P2, which has no DnaA requirement, yielded a mutant whose locus we mapped to the rep gene. A 186 mutant able to infect this rep mutant was isolated, and the mutation was located in the phage replication initiation endonuclease gene A, suggesting direct interaction between the Rep helicase and phage endonuclease during replication. DNA sequencing indicated a glutamic acid-to-valine change at residue 155 of the 694-residue product of gene A. In the discussion, we speculate that the indirect need of DnaA function is at the level of lagging-strand synthesis in the rolling circle replication of 186.

L8 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2003 ACS
AN 1994:625918 CAPLUS
DN 121:225918
TI Replication mechanisms of plant RNA viruses
AU Ishikawa, Masayuki
CS Faculty Agriculture, Hokkaido University, Kita, 060, Japan

SO Uirusu (1994), 44(1), 3-10
CODEN: UIRUAF; ISSN: 0042-6857
DT Journal; General Review
LA Japanese
AB A review with 52 listed refs. on the **replication** mechanism of tobacco mosaic **virus** (TMV). It comprises termination of accumulation of minus-strand RNA in the beginning of infection, and the **screening** of the factors of **host** that are involved in the replication of TMV.

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